

SEVERE PERIPROSTHETIC CORTICAL ATROPHY IN THE SKELETALLY IMMATURE: A REPORT OF THREE CASES

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ABSTRACT

We describe three patients who developed cortical bone atrophy around cemented endoprostheses used for partial femur reconstructions after resection of Ewing's sarcoma. We believe this to be related to remodeling secondary to stress-shielding. Rather than increased porosity and decreased mineral density, the stress-shielding in these skeletally immature patients resulted in altered morphology of the cortical bone, with apparent maintenance of density.

INTRODUCTION

Stress-shielding is a well-recognized phenomenon associated with many clinical scenarios in which an implant with higher elastic modulus than bone bears the majority of stress transmitted through an anatomic region by fixation to bone above and below. This prevents the load bearing necessary for osseous homeostasis, and thus encourages a disuse-type osteopenia in the accompanying bone. It is most widely discussed in relation to diaphyseal fit designs for noncemented total hip arthroplasty, but is recognized in many other scenarios including external and internal fixation for fractures and cemented hip arthroplasty. Stress-shielding usually results in osteopenia, or an atrophy of bone mineral and trabecular density, rather than an atrophy of bone morphology overall.

We present three skeletally immature patients who developed peri-implant cortical atrophy following endoprosthetic reconstructions for bone defects created by resection of malignant neoplasms.

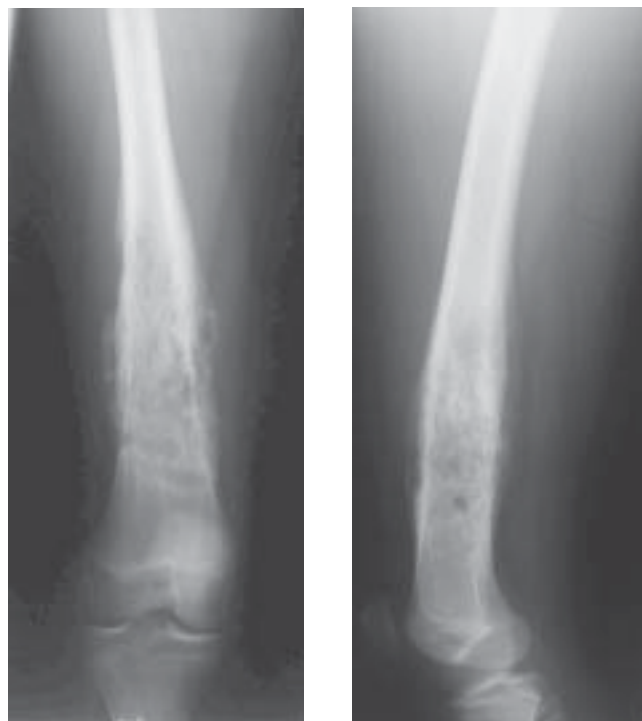


Figure 1. AP and lateral radiographs of the distal femur in case 1, a 13-year-old female with Ewing's sarcoma.

PATIENTS

Case 1

The first patient was diagnosed with a Ewing's sarcoma of the distal femur at 13 years of age (Figure 1). She was treated with neo-adjuvant and adjuvant chemotherapy, resection and cemented endoprosthetic reconstruction of her distal femur and knee (Figures 2A and 2B). She developed no recurrence and returned to excellent function after reconstruction. Over time, however, the periprosthetic cortical bone atrophied (Figures 2C and 2D). The patient continues to have excellent knee function without fracture or failure, eight years after her resection. She has declined repeated encouragements to consider prophylactic onlay reinforcement of the atrophied implant-bone junction.

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Figure 2A



Figure 2B



Figure 2C



Figure 2D

Figure 2. AP and lateral radiographs of the distal femoral endoprosthesis reconstruction after resection of a Ewing's sarcoma (case 1). Panels A and B were obtained one month after resection and reconstruction; panels C and D were obtained eight years after reconstruction.



Figure 3. AP and lateral hip radiographs demonstrating the pathologic proximal femur fracture through a Ewing's sarcoma in a ten-year-old (case 2).



Case 2

The second patient presented at 10 years of age with a pathologic subtrochanteric femur fracture through a Ewing's sarcoma (Figure 3). The fracture punctuated a two-month history of thigh pain treated conservatively for a presumptive diagnosis of Osgood-Schlatter after knee imaging showed no abnormality.

An open biopsy was obtained and the fracture internally fixed with a pediatric hip screw prior to neoadjuvant chemotherapy (Figure 4). Her resection demonstrated negative mar-



Figure 4. AP radiographs demonstrating the reduced and internally fixed pathologic fracture after biopsy confirming Ewing's sarcoma in a ten-year-old (case 2).



Figure 5A



Figure 5B



Figure 5C



Figure 5D

Figure 5. AP and lateral radiographs of the proximal femur shortly following (A and B) and three years after (C and D) resection of a Ewing's sarcoma and reconstruction with a proximal femur-replacing endoprosthesis.

gins and excellent necrosis response to chemotherapy. She was also reconstructed with a cemented modular endoprosthesis (Figures 5A and 5B). Adjuvant chemotherapy was completed uneventfully. Treatments included Idamycin, Cytosan, VP16, ifosfamide, and anthracycline.

Other than the anticipated limb-length discrepancy that developed with growth, the only concern that arose after full functional recovery was progressive and severe cortical atrophy (Figures 5C and 5D). She was first advised to undergo onlay strut allograft reinforcement of the bone-implant junction at two-and-one-half years after her resection and reconstruction. She agreed to proceed with this another year later (Figure 6). She continues to do well, now 18 months after the reinforcement surgery.



Figure 6. AP and lateral radiographs of the proximal femur demonstrating onlay allograft strut reinforcement of the prosthesis-bone junction, performed due to concern for severe periprosthetic cortical atrophy.



Figure 7. AP and lateral radiographs of the proximal femur demonstrating a Ewing's sarcoma of the trochanteric region and femoral neck in an eight-year-old female.

Case 3

The final patient presented at eight years of age with thigh pain and systemic illness. She was diagnosed with a proximal femoral Ewing's sarcoma and associated pulmonary metastatic disease (Figure 7). Treatment for the sarcoma included chemotherapy and radiation to both femur and thorax. A total of 5580 centi-Gray units (cGy) of external-beam radiation was administered to the proximal femur. Fifteen months later, she sustained a fracture through the irradiated bone, which was treated with pediatric hip screw prophylactic fixation (Figures 8A and 8B).

Five otherwise-uneventful years after the initial diagnosis, she developed a chondroblastic osteosarcoma in the previously irradiated proximal femur (Figures 8C and 8D). This was treated with neo-adjuvant and adju-

vant chemotherapy, wide resection and cemented endoprosthetic proximal femoral reconstruction (Figures 9A and 9B).

Beginning approximately three years after her reconstruction, cortical atrophy at the implant-bone junction progressed to a worrisome degree. She was advised to consider onlay strut allograft reinforcement of the bone-implant junction but declined such an intervention.

At six years after her initial reconstruction, she sustained a catastrophic failure from minor trauma, sustaining a periprosthetic femur fracture and prosthetic stem fracture (Figures 9C and 9D). This was successfully treated with a revision modular hip prosthesis (Figure 10). She continues to do well, despite concern for insufficiently robust bone at the implant junction, now three years later.



Figure 8A

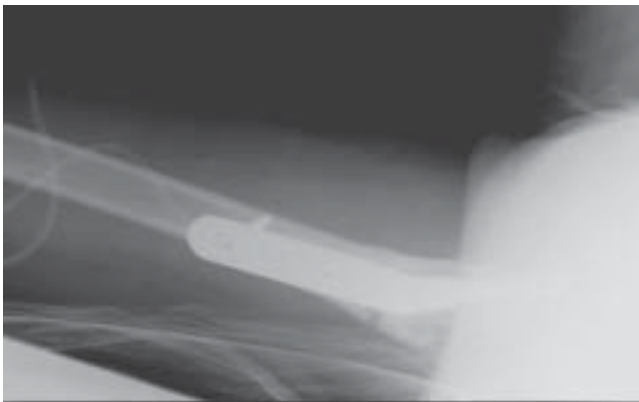


Figure 8B



Figure 8C



Figure 8D

Figure 8. AP and lateral radiographs (A and B) after open reduction and internal fixation of a subtrochanteric fracture that was sustained 15 months after diagnosis of Ewing's sarcoma, treated with external beam radiation. Panels C and D demonstrate the chondroblastic osteosarcoma that subsequently developed in the same region, another three-and-one-half years later.



Figure 9A



Figure 9B



Figure 9C



Figure 9D

Figure 9. AP and lateral radiographs demonstrating endoprosthesis reconstruction of the proximal femur after resection of a post-radiation chondroblastic osteosarcoma. Panels A and B represent early post-operative radiographs; C and D represent a periprosthetic fracture with prosthetic stem fracture sustained six years later.

DISCUSSION

Charnley first described the phenomenon of stress-shielding in his 1968 mid-term follow-up of 190 of his early cemented hip arthroplasties¹. Stress-shielding was manifested in two radiographic features. Over one-third of his patients had radiographic resorption of two-to-three millimeters of the calcar femorale, separating it from the collar of the prosthesis and its initial abutting position. Second, just under five percent of his patients had developed greater than five percent relative atrophy of the peri-implant cortex. As understanding of stress-shielding increased over the years, it came to be specifically associated with loss of bone mineral density in the trochanteric region proximal to a well-fixed diaphyseal fit non-cemented prosthesis. It is interesting, however, that in Charnley's original description of the phenomenon, he used cortical thinning as his measure.

We reviewed three cases of significant peri-prosthetic cortical atrophy of the femoral diaphysis following cemented reconstruction with a modular tumor prosthesis in two adolescents and one pre-adolescent. The etiology remains uncertain, but excessive remodeling from stress-shielding is suspected.

Tumor recurrence should always be primarily ruled out when any osseous changes are noted after resection of a malignant neoplasm. This was considered carefully in each case. That it was appropriately ruled-out is evidenced by the continued event-free survival of all of the patients, years after recognition of the atrophy.

The magnitude of the stress-shielded remodeling response may be due to side effects of chemotherapy or even radiation (in case three, only), but the fact that the morphology of the cortical bone changed rather than porosity, argues that neither osteoblasts nor osteoclasts were relatively impaired. It is interesting that all three of the patients had Ewing's as a primary diagnosis, but this yields no obvious explanation of bone changes after disease eradication. Mostly likely, the well-recognized increased osteoclastic response to minimal load bearing secondary to stress-shielding is better matched in these skeletally immature patients by osteoblastic recontouring of the shape of the cortical bone.

Periprosthetic bone remodeling has previously been studied by the oncology group in Toronto for distal femoral replacements with a noncemented Kotz prosthesis after resection of malignancies². They demon-



Figure 10A



Figure 10C



Figure 10D

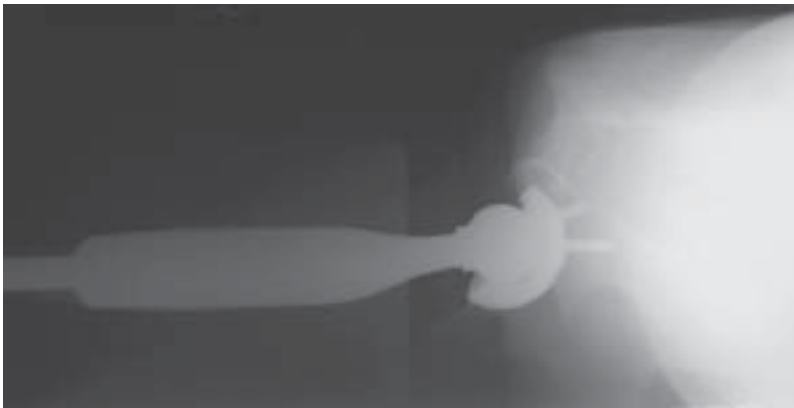


Figure 10B

Figure 10. AP and lateral radiographs of the proximal and distal femur demonstrating the revision endoprosthesis reconstruction undertaken to treat a periprosthetic fracture from a previous implant. Cortical atrophy around the new implant remains a concern.

strated that bone mineral density loss as measured by DEXA averaged 23.7 percent in the region immediately adjacent to the base of the prosthetic stem.

Whatever the explanation, cortical atrophy surrounding a modular implant increases a patient's risk for catastrophic failure of the implant. Such events require urgent, major surgery rather than the elective circumstances possible for prophylactic reinforcement. It will continue to be our practice to look for these changes radiographically and recommend prophylactic onlay strut allograft reinforcement when such atrophy is recognized.

REFERENCES

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